| Ammonia | |
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| **Description** | Circulating ammonia is produced principally from the catabolism of amino acids derived from endogenous or dietary proteins. The gastrointestinal tract is an important source. Usually, plasma concentrations are maintained at low levels by urea synthesis in the liver and by formation of glutamine. |
| **Indication** | For the investigation of encephalopathy and unexplained neurological symptoms in babies and the elderly. |
| **Additional Info** | Contamination of the sample by ammonia from exogenous sources or due to endogenous production during specimen transport and handling are the most common causes of inaccurate results. Smokers usually have higher plasma ammonia concentrations. Haemoglobin does not interfere with ammonia determinations, but the ammonia concentration within erythrocytes is about three times the concentration in plasma, so haemolyzed samples are unsuitable. |
| **Concurrent Tests** | N/A |
| **Dietary Requirements** | Fasting is recommended but not necessary in some situations e.g. ITU patients. |
| **Interpretation** | Hyperammonaemia occurs when there is disruption of the urea cycle in the liver. This may be due to inherited defects of the urea cycle enzymes secondary to disruption of liver metabolism or bacterial production of ammonia in the gastrointestinal or urinary tract. The urea cycle defects are rare disorders but have an estimated combined incidence of about 1:30,000, with the commonest being ornithine transcarbamylase deficiency (OTC). Some disorders of amino acid, organic acid, and fatty acid metabolism may also present with hyperammonaemia. In neonates, clinically significant hyperammonaemia may occur secondary to asphyxia, infection, and sepsis. Hyperammonaemia has been associated with parenteral nutrition, sodium valproate therapy, acute-onset hepatic failure, and advanced chronic liver disease. In Reye's syndrome, acute encephalopathy and fatty degeneration of the liver may be associated with a viral illness or salicylate therapy that lead to acute mitochondrial injury. Ammonia is neurotoxic. Hyperammonaemia has a range of presentations from an acute catastrophic illness in babies to episodes of lethargy and vomiting. Measurement of ammonia should be considered in any neonate with unexplained neurological deterioration, any older patient with unexplained encephalopathy, and children or adults with a history of episodes of vomiting and lethargy or of protein avoidance, which may indicate a mild urea-cycle defect. Increased ammonia concentration should always be confirmed on a second sample to exclude artifactual increases caused by poor sample handling. Infection should always be excluded as a cause of mild increases in ammonia in neonates. Inherited disorders should be excluded in patients with unexplained hyperammonaemia, Reye's-like syndrome, unexplained hepatic encephalopathy, or cyclical illness. Increased ammonia concentrations are a nonspecific finding in liver disease, and measurements do not aid with diagnosis, prognosis, or monitoring of treatment. Significant encephalopathy usually develops at concentrations above 300 µmol/L. Concentrations greater than 500 µmol/L usually present with coma and convulsions and are associated with neonatal-onset inherited metabolic diseases. |
| **Collection Conditions** | Venous samples collected in K-EDTA without stasis and avoiding |

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| | haemolysis. Blood collection tubes should be filled completely, place on ice and the blood sample sent to the lab immediately. |
| **Frequency of testing** | N/A |